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EDITORIAL COMMENT

Straining the RV to Predict the Future*

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It would seem that the "oft forgotten ventricle" can be forgotten no longer. For many decades, we have looked to the left ventricle (LV) as the major determinant of a patient's cardiac health and prognosis, but a study in this issue of *iJACC* by Park et al. (1) reminds us that the right ventricle (RV) is an important predictor of clinical outcomes and possibly the most important. It is not surprising that RV function is a critical determinant of survival in pathologies of the pulmonary vasculature, such as pulmonary arterial hypertension (2,3). However, RV function is also an important prognostic marker in conditions that have traditionally been regarded as primarily LV pathologies such as congestive heart failure (4-6) and acute myocardial infarction (7-9). Whether measured with echocardiographic M-mode, 2-dimensional (2D), newer strain techniques, or magnetic resonance imaging, the conclusions are concordant; RV function can be reliably assessed and is a critical marker of clinical outcomes even after clinical and LV parameters have been taken into account.

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Why does the RV provide such a useful window for the prediction of cardiac outcomes? As summarized in **Figure 1**, RV function reflects, not only the intrinsic myocardial contractility of the RV, but also the afterload effect imposed by increases in LV filling pressures and pulmonary vascular pathology. As compared with the LV, RV function is more profoundly affected by increases in afterload (10), and therefore, the RV becomes a very sensitive barometer of any "downstream" factors. In the post-myocardial infarction setting, echocardiographic estimates of pulmonary artery pressures provide important prognostic information (11), most likely as a consequence of raised LV filling pressures. The combination of raised pulmonary pressures and RV dysfunction suggests that the contractile reserve is struggling to maintain output against the heightened afterload and portends a particularly ominous prognosis (2,12). Overall cardiac output is "only as good as the worst ventricle," and Park et al. (1) provide yet more evidence that the RV can represent a weak link following myocardial infarction. However, a more comprehensive assessment including afterload estimates would have enabled Park et al. to provide greater insights into whether the predominant pathology is contractile impairment of the RV, heightened afterload as a result of LV failure, or a combination of both.

Most previous studies have used simple measures such as RV fractional area change or tricuspid annular plane excursion to demonstrate the prognostic importance of RV function in patients with heart failure or recent myocardial infarction. As an important advance, Park et al. (1) compare these established measures with newer 2D deformation measures and conclude that RV longitudinal strain provides better predictive accuracy. They used velocity vector imaging, a technique that tracks unique 2D ultrasound patterns within the RV endocardium and measures the distance and direction of their movement between image frames. This is similar, but not identical, to other 2D strain techniques, and the degree to which the results of this study can be generalized remains untested (13). Furthermore, deformation of the interventricular septum is predominantly affected by the LV, and there is contention over whether it should be incorporated in global measures of RV function. The data of Park et al. (1) suggest that these unresolved issues may be of limited importance. They demonstrated that all measures of RV function predicted cardiac events, and thus we may conclude that quibbling over which is the best measure should not obscure the importance of measuring the RV with something. However, if all tools are available, then 2D strain represents a

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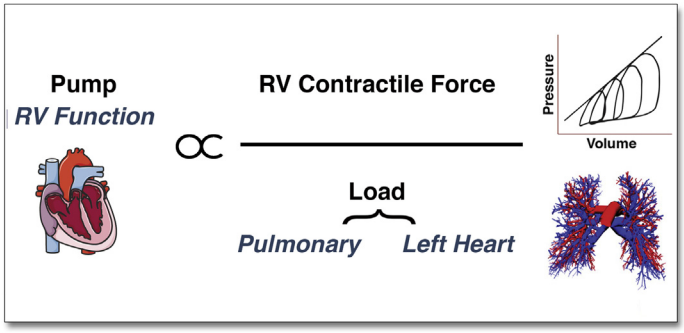
simple and efficient measure that may provide a better summary of global RV function than traditional measures.

Does the association between RV dysfunction and worse outcomes matter? Can this information be used to improve clinical outcomes? The cynic might argue that there are no specific RV treatments and so these findings are largely academic. However, it is important to note that improvements in LV function and LV loading impact favorably on the RV, both by means of direct ventricular interaction and by the fact that reductions in LV filling pressures will decrease the pulmonary afterload. Thus, RV function may be a marker of treatment efficacy that provides the clinician with a more accurate means of targeting those patients in whom more aggressive therapy may be required. For example, RV measures are not currently considered in patient selection algorithms for defibrillator or synchronization device therapies. There is no evidence to support this practice, but given that RV dysfunction is so clearly associated with worse patient outcomes, it would seem a logical hypothesis to aim more aggressive treatment at these patients. Furthermore, with some evolving optimism that pulmonary vasodilators may have a role to play in the treatment of heart failure (14), successful therapy may be dependent on selecting patients with RV impairment.

There have been massive advances in the past decades in the management of myocardial infarction and heart failure. It may be that further significant advances are going to require better selection of treatments for specific patients. The study of Park et al. (1) contributes to an exciting evolving field in which we are getting better at quantifying RV dysfunction and identifying at-risk patients. Therapeutic decisions influenced by RV strain measurements may not be so far away.

FIGURE 1 Relationship Between RV Function, Contractile Force, and Load



Right ventricular (RV) function is proportional to its contractile force and inversely related to its afterload. RV afterload is determined by the filling pressures of the left ventricle and the resistance of the pulmonary circulation.

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